Take Home Worksheet: Respiration Challenge

All of the following questions are the same or similar to ones discussed in class today. One good strategy for working with these questions is to answer them on your own to the best of your ability, then compare your answers with a fellow student. Together you will be able to create good answers to the questions. In all cases, be prepared to explain your reasoning clearly and succinctly.

1. In the 1940s, some physicians prescribed the drug DNP (dinitrophenol) to help patients lose weight. This treatment was abandoned after several patients died. DNP works by uncoupling the chemiosmotic machinery by making the lipid bilayer of the inner mitochondrial membrane leaky to H\(^+\). A) Explain how DNP causes weight loss. B) Suggest at least one other cellular effect of DNP (i.e., what other aspects of the cell might be affected by DNP), and prepare an explanation of this effect.

2. Cyanide is a chemical that irreversibly binds to (i.e., prevents the functioning of) the enzyme cytochrome oxidase, an important enzyme in the electron transport system. A) Explain, at the cellular level, why cyanide is a lethal chemical. B) Suggest at least one other cellular consequence of cyanide (i.e., what else happens to the electron transport system when cyanide is present), and prepare an explanation of this effect.

3. Rotenone is a naturally occurring chemical derived from the roots of several tropical and subtropical plants. Contact, particularly ingestion, with rotenone can be fatal. It inhibits chemiosmosis by interfering with NADH dehydrogenase preventing utilization of NADH as a proton and electron donor in the electron transport chain. A) Explain why rotenone exposure can be fatal. B) Suggest at least one other cellular effect of rotenone (i.e., what other parts of respiration might be affected by rotenone), and prepare an explanation of this effect.

4. DCCD (dicyclohexylcarbodiimide) inhibits oxidative phosphorylation when the substrate is mitochondrial NADH. DCCD is a drug that binds to ATP synthase and blocks proton transport through the ion channel. A) Explain what the consequences are of DCCD on cellular energy production. B) Suggest at least one other cellular effect of DCCD, and prepare an explanation of this effect.

5. Sometimes the supply of oxygen in active muscle tissue is not adequate for the demands of oxidative phosphorylation. When this situation occurs, the electron transport system slows. A) Explain on a cellular level the consequences of low oxygen supply. B) Suggest a respiration-based explanation for why deep breathing continues even after strenuous exercise has stopped.

6. How much energy would be generated in the cells of a person who consumed a diet of pyruvate instead of glucose? Contrast the energy production of a high carbohydrate diet and a high protein diet.

7. Do mitochondria raise or lower the pH of the region of the cell in which they are found? Why? Describe the relative pHs of the cytoplasm, the mitochondrial intermembrane space, and the mitochondrial matrix, with an explanation of the origin of this pattern.

8. Glucosamine is a common compound that inhibits hexokinase action (the transfer of a phosphate group to glucose during glycolysis). A) Describe the consequences of
glucosamine exposure on cellular respiration. B) Is glucosamine exposure fatal? Why or why not?

9. Vitamin B3 (niacin) is a component of NAD+ (or NADH). Niacin is acquired through the diet. A) Describe the consequences of niacin deficiency on energy production. B) Invent two strategies a cell might use to maintain energy production under niacin deficiency.

10. Anemia is characterized by low hemoglobin levels or a reduced number of red blood cells (both of which transport oxygen to cells). A common symptom of anemia is tiredness. A) Explain what the consequences are of anemia on cellular energy production. B) Suggest at least one other cellular effect of anemia, and prepare an explanation of this effect.

11. Antimycin A is a pesticide in use worldwide. It is recognized as a respiration inhibitor, since it blocks the electron transport chain between cytochrome b and cytochrome c₁. A) Describe why antimycin A is a successful pesticide. B) Describe the effects of antimycin A exposure on cellular respiration in terms of the byproducts of the process.

12. Bongkrekic acid is a toxic compound produced by the bacteria species Burkholderia gladioli. If ingested, bongkrekic acid most often results in death. Bongkrekic acid inhibits the adenine nucleotide transporter that shuttles ADP across the inner mitochondrial membrane. A) Describe the consequences of bongkrekic acid poisoning on cellular respiration. B) Suggest two chemical cues that might indicate bongkrekic acid poisoning.

13. Oligomycin is an antibiotic compound produced by actinomycete bacteria (these bacteria can cause the condition known as lumpy jaw). Oligomycin binds to a protein in the ATP synthase complex. A) Describe the consequences of oligomycin on cellular respiration. B) Describe the energy production capacity of a cell affected by oligomycin.

14. The antibiotic valinomycin is one example of an ionophore, a chemical that makes the inner mitochondrial membrane permeable to protons. A) Describe why valinomycin is an effective antibiotic agent. B) Suggest three biochemical consequences of valinomycin on cellular respiration.